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The Kindness of Psychopaths

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ABSTRACT

Psychopathy attracts considerable interdisciplinary interest. The idea of a group of people with abnormal morality and interpersonal relations raises important philosophical, legal and clinical issues. However, before engaging these issues, we ought to examine whether this category is scientifically grounded. We frame the issue in terms of the question whether ‘psychopathy’ designates a natural kind according to the cluster approaches. We argue that currently there is no sufficient evidence for an affirmative answer to this question. Furthermore, we examine three ways of dealing with the category of psychopathy. We could eliminate the category, revise it, or subscribe to a more encompassing account of kinds, which could capture psychopathy as it is currently conceptualised. We argue that while a revision of the category of psychopathy is to be expected with empirical and theoretical advancements, we also emphasise its role in clinical and forensic research, which makes it an important pragmatic kind.

1. Introduction

Psychopathy is a personality disorder that most notably involves a diminished sense of guilt, remorse and empathy. Other characterisations involve fearlessness, impulsivity and proneness to risky and gratifying behaviour. In addition, the diagnostic criteria for psychopathy include failure to make long-term plans, behaving irresponsibly and disregard for social norms (Hare 2003). In recent decades, we are witnessing an increasing interest in the scientific study of psychopathy (Blair, Mitchell, and Blair 2005; Patrick 2006; Glenn and Raine 2014) as well as investigations on the appropriate social response to psychopathic offenders (e.g. Malatesti and McMillan 2010; Caldwell and Van Rybroek 2013).

The classification of certain individuals as psychopaths is at the core of important scientific and related normative discussions. Whether the construct of psychopathy picks out a unified phenomenon is a matter of substantial theoretical and empirical debate among scientists who are investigating this condition (Skeem et al. 2011; Brazil et al. 2016). Moreover, psychopathy has figured prominently in many recent philosophical and applied debates. For instance, some authors have argued that the existence of psychopaths might have implications for the nature of moral judgement and other

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issues in metaethics (Prinz 2006; Sinnott-Armstrong 2014; Kumar 2016). As for the practical repercussions, some authors have argued that scientific research warrants diminished legal or moral accountability for individuals falling under the category of psychopathy (Malatesti and McMillan 2010; see also Jalava and Griffiths 2017). Furthermore, some have argued that psychopathy should be considered as a mental illness (Nadelhoffer and Sinnott-Armstrong 2013) or even that psychopaths should not be included as full-fledged members (with all the relevant moral and legal rights) into our society (e.g. Gaus 2011, 210, 282–283).

Arguably, these debates are not explicitly committed to the view that psychopathy is a natural kind. Still, the question whether psychopathy is a natural kind bears weight on these debates, because, in one way or another, they presuppose that psychopathy presents a sufficiently unified phenomenon warranting sound inductive generalisations about people so classified. In fact, despite mixed evidence on the unity of the construct ‘in most clinical and legal contexts psychopathy is instead construed and assessed as if it were a single thing: a homogeneous diagnostic category underpinned by a single causal process’ (Skeem et al. 2011, 115).

In this article, we proceed as follows: in the next section, we introduce cluster approaches to natural kinds. In section 3, we offer a more elaborate description of the category of psychopathy and some prominent measures of it. In section 4, we evaluate the hypothesis that psychopathy might be a natural kind on the homeostatic property cluster (HPC) and the stable property cluster (SPC) accounts. We examine the claim that psychopathy is a syndrome comprised of highly correlated traits and argue that, despite there being studies indicating that psychopathic traits significantly correlate, there is insufficient evidence that they comprise an SPC kind. In addition, we consider whether psychopathy is underpinned by a characteristic causal aetiology. We argue that the lack of success in establishing specific and well-delineated aetiology or biomarkers for psychopathy indicates that psychopathy in the current stage of research cannot be considered an HPC kind. Thus, we argue that the construct of psychopathy denotes a heterogeneous category that has limited theoretical utility.

In section 5, we investigate what is the appropriate methodological response to such a situation. We examine three options: (1) revising the category of psychopathy; (2) eliminating it; or (3) abandoning the cluster approach to natural kinds. We argue against the last two options. We argue that option (1) is likely to advance future research on psychopathy. However, in section 6, we indicate that the need for revision of the category does not necessarily entail that the category ought to be eliminated, at least not until a suitable alternative becomes available. We offer a more general discussion of how to accommodate the current state of psychopathy research. The category of psychopathy is at least pragmatically important because it provides a framework for clinical and forensic research. Thus, we indicate how treating psychopathy as a *pragmatic kind* can account for its status at the current stage of research.

2. Natural Kinds in the Biomedical Sciences and Psychiatry

Essentialism is a traditional approach to natural kinds—that conceives them as groupings of things that share a set of common characteristic properties (essences). The essence is supposed to be a necessary and sufficient condition for kind membership. However, essentialism about natural kinds is inadequate as a description of kinds in many disciplines,

especially, in the biomedical sciences, where strict divisions into categories as required by essentialism are often impossible (Dupré 1993).

One possible reaction is to conclude that there are no natural kinds in the biomedical and social sciences. However, the downside of this approach is that it appears to be utterly uninformative; in fact, disciplines, such as psychiatry, utilise classifications and the question what makes some of those classifications better, i.e. more successful, closer to the 'real' groupings of properties in nature, seems to be a valid one (Murphy 2006, ch. 7). In fact, many philosophers of psychiatry agree that real mental disorders are natural kinds (Beebe and Sabbarton-Leary 2010). For instance, Richard Samuels (2009) argues that delusions, while Jonathan Tsou (2013, 2016) argues that schizophrenia, depression, and even suicide are natural kinds.

However, these authors usually adopt a more inclusive view of natural kinds. According to an influential account, natural kinds are clusters of co-occurring properties unified by causal homeostatic mechanisms (Boyd 1991, 1999). These mechanisms are responsible for the clustering of properties that ground inductive generalisations about kind members. In the philosophy of science, properties that ground inductive inferences are called projectible properties. These properties, in turn, enable us to systematically classify and predict the behaviour of kind members. One important distinction is between the mechanisms and causes that are intrinsic and those that are extrinsic to the members of the kind. For instance, in the case of biological species, extrinsic mechanisms or causes such as interbreeding, common ancestry and shared ecological niche are important, because they cause shared intrinsic, i.e. genetic and developmental properties that are responsible for the observable phenotypic traits. Inclusion of extrinsic or relational mechanisms or properties is a novelty introduced by HPC, which makes it suitable for classifications in the life sciences.

The search for homeostatic mechanisms can be related to the search for aetiologies of different disorders or conditions in psychiatry and related fields. In this respect, there are various levels and types of explanation of interest (Kendler, Zachar, and Craver 2011). One type of explanation may invoke distal factors that go back in evolutionary history, or more proximal causes such as genetic and/or environmental processes underlying certain disorders. When we talk about shared intrinsic properties, we invoke brain states as the best candidate for the scientific grounding of certain behaviours and actions. Depending on how many of these features are shared and how much we know about the underpinning mechanisms, our explanations and predictions invoking such categories will have a higher or lower degree of reliability.

Many philosophers of psychiatry have endorsed the HPC account as suitable for accommodating psychiatric categories (e.g. Samuels 2009; Beebe and Sabbarton-Leary 2010; Kendler, Zachar, and Craver 2011). A reason for HPC's popularity is that it captures well the actual practice of classification across sciences. Boyd's view is also widely endorsed in the more general debates on classification in the philosophy of science (Ereshefsky and Reydon 2015), and is applied in many scientific areas (e.g. Feldman Barrett 2006; Tsou 2013; Murphy 2014).

There are, however, several objections to the HPC account. For instance, Muhammad Khalidi (2013) claims that the demand for underlying homeostatic *mechanisms* is too strong. Nevertheless, he maintains that a causal account of natural kinds is a correct one, where some common causes account for the similarities of kind members.

Matthew Slater (2015) further argues that it is enough that there are clusters of *stable properties* (SPC), and that we should not worry about the underlying causes of such stability. On this view, roughly, clusters of properties are stable if instantiation of some of them warrants a probabilistically reliable inference to the instantiation of other properties (Slater 2015, sec. 5; see also note 8 below). In our case, this would mean that psychopathy is a kind if observations of some psychopathic traits probabilistically entail the presence of others. Unfortunately, Slater does not provide reliability thresholds for probabilistic entailments that could be directly applied to the classification of psychopathy. He claims that depending on a scientific field, this might be contextually determined (Slater 2015, n. 29). In section 4, we indicate what would ground a reliable inference relevant for our discussion.

Here, we do not adjudicate between the proposed accounts of natural kinds. We maintain that clustering accounts of natural kinds (whether it is HPC or the more encompassing SPC account) are appropriate for psychiatric categorisations (Kendler, Zachar, and Craver 2011; Murphy 2014). Furthermore, HPC seems to account for the way in which many researchers conceptualise the construct of psychopathy (see section 4 here). We acknowledge, however, that there are useful and interesting examples of psychiatric categories that cannot be captured by the clustering accounts, as will be shown with the case of psychopathy (for a taxonomy of classifications in psychiatry, see Haslam 2002). With regard to such cases, it appears that a reconceptualisation of the category is in order, but the question remains how to proceed in the scientific inquiry before a better alternative gains prominence. In the case of psychopathy, we propose to conceptualise it as a pragmatic kind.

In the next section, we introduce in more detail the construct of psychopathy. Then in sections 4 and 5, we discuss whether psychopathy as currently conceptualised should be considered a natural kind according to cluster views.

3. The Psychopathy Construct

As already noted, many researchers take psychopathy to be a scientifically robust concept since it denotes a set of traits consistently found across individuals, cultures and ethnic groups (Cooke 1998). Psychopathy is associated with extreme antisocial behaviour and characteristic emotional and interpersonal traits (Table 1). These traits distinguish it

Table 1. PCL-R items (Hare 2003).

Factor 1	Factor 2
Facet 1: Interpersonal traits 1. Glibness/Superficial charm 2. Grandiose sense of self-worth 4. Pathological lying 5. Conning/Manipulative	Facet 3: Lifestyle traits 3. Need for stimulation 9. Parasitic lifestyle 13. Lack of realistic, long-term goals 14. Impulsivity 15. Irresponsibility
Facet 2: Affective traits 6. Lack of remorse or guilt 7. Shallow affect 8. Callous/Lack of empathy 16. Failure to accept responsibility	Facet 4: Antisocial traits 10. Poor behavioural controls 12. Early behavioural problems 18. Juvenile delinquency 19. Revocation of conditional release 20. Criminal versatility
Items not belonging to any of the facets: 11. Promiscuous sexual behaviour, 17. Many short-term marital relationships	

from a more general Antisocial Personality Disorder (ASPD) as registered in different editions of the Diagnostic-Statistical Manual (DSM). In the latest (5th) edition of DSM (American Psychiatric Association 2013, DSM V), however, ASPD has been revised and now it is also defined by interpersonal and affective traits, which makes it closer to contemporary conceptualisations of psychopathy (Strickland et al. 2013). Nevertheless, psychopathy is still not explicitly recognised as a personality disorder in DSM V, and thus we should be careful not to conflate these two constructs (Brazil and Cima 2016).

Modern conceptualisations of psychopathy are largely based on Hervey Cleckley's (1976) seminal work. He made a list of psychopathic traits based on his clinical practice and interviews with different types of disturbed individuals. In his book, *The Mask of Sanity*, he provides a 'clinical profile' of a typical psychopath. Robert Hare (2003) introduced the Psychopathy checklist (PCL and the later revised versions PCL-R, see Table 1) to operationalise Checkley's 'clinical profile' for research. PCL-R is widely used for diagnosing psychopathy in prison and other institutionalised settings. It also serves as a unifying tool for empirical, theoretical and practical research on psychopathy (Mala-testi and McMillan 2014). As such, it is regarded as the 'gold standard' for psychopathy research.

By using PCL-R, individuals are assessed on 20 items via semi-structured interviews and intensive study of their history (mostly, the prison records).¹ Scores range from 0 to 2. The cut-off value for being a psychopath is 30 in North America, while in Europe, it is 25.² However, the appropriate cut-score is pragmatically determined. For instance, if it has practical consequences for a person involved (e.g. risk assessment or treatment options) then Hare suggests using higher cut-scores, while for research purposes he suggests that using lower scores might be appropriate (Hare 2003, 30–31).

Early statistical analyses of PCL-R indicated that some items are more correlated than others. For instance, people scoring high on pathological lying also score high on the conning/manipulative item. Similarly, scoring high on lack of remorse is correlated with scoring high on callous/lack of empathy. Factors in statistical analysis represent groupings of items that are significantly correlated with each other. Based on such correlations, Hare (2003) accepts the division of PCL-R items into two factors, which further divide into 4 facets (Table 1). Factor 1 groups together interpersonal and affective traits, while Factor 2 captures lifestyle and antisocial traits.

The virtues of the PCL-R also hinder its application to general populations.³ PCL-R relies on accessing files of subjects' histories and extensive interviews, which are not always available, especially when studying non-forensic populations. Thus, with general populations, alternative measures are often used.

One widely used tool is the Psychopathic Personality Inventory (PPI; Fowler and Lilienfeld 2013). The PPI is a self-report measure originally consisting of 187 items. Similarly, to PCL-R, the items can be divided into two broad factors. Fearlessness-dominance (FD) factor includes Social Potency, Fearlessness, and Stress Immunity. These include traits such as absence of anxiety, willingness to take risks, and skill at influencing others. Antisocial-impulsivity (AI) factor comprises Machiavellian Egocentricity, Impulsive Nonconformity, Blame Externalisation, and Carefree Nonplanfulness. These include traits such as lack of concern for the social norms, attitude of indifference towards future, and 'a ruthless willingness to manipulate and take advantage of others' (Fowler and Lilienfeld 2013, 44). Some psychometric analyses have indicated convergent

validity between PPI and PCL-R (Poythress et al. 2010, 10). Thus, it is thought that FD corresponds to Factor 1, while AI corresponds to Factor 2 of PCL-R.

4. Is Psychopathy a Natural Kind?

Although, as far as we are aware, ‘natural kinds’ language is not used in the psychopathy research, an important group of authors accepts that psychopathy satisfies the cluster approach criteria for kindhood. In psychology and the biomedical sciences, the notion of syndrome is usually used to designate a constellation of correlated symptoms. When the symptoms are highly correlated, it is thought that they are underpinned by a common cause. If a condition is comprised of weakly correlated symptoms, then it is assumed that the symptoms stem from heterogeneous causes (Feldman Barrett 2006, 33; Lilienfeld 2013, 85). In the vocabulary of natural kinds, a constellation of highly correlated symptoms would correspond to a projectible property cluster, and the assumption of common aetiology to a sustaining cause. Many psychopathy research groups think of psychopathy as a syndrome (for a review, see Hamilton, Racer, and Newman 2015). As a paradigmatic example, James Blair and colleagues claim that psychopathy comprises a projectible overlapping cluster of emotional and interpersonal traits underpinned by specific neurocognitive mechanisms (Blair, Mitchell, and Blair 2005, 7, 15).

In what follows, first, we examine whether psychopathy is a syndrome comprised of highly correlated traits. Secondly, we survey two main alternative hypotheses regarding the underlying causes of psychopathy. Thirdly, we discuss attempts to delineate psychopathy with neurobiological markers.

4.1. Psychopathy as a Stable Property Cluster

Psychopathic personality traits are relatively stable across time (Glenn and Raine 2014, 23), but this temporal stability is not sufficient for considering it a natural kind. On the SPC account, stability refers to reliable inferences from observations of certain features to instantiations of other features (Slater 2015). In the psychological research, this claim translates into correlations. Namely, the higher the correlation between traits, the higher the probability that the traits are co-instantiated. A high correlation indicates the existence of a discrete category or taxon. Thus, according to Feldman Barret, a ‘way to establish the presence of an abstract construct ... is to demonstrate that [it] has measurable effects that are highly correlated’ (Feldman Barrett 2006, 33). In our case, if psychopathy is a natural kind, its measurable behavioural, affective and interpersonal traits should highly correlate and form a taxon (Skeem et al. 2011, 101–102).

Although some authors have argued that psychopathy is a taxon, specifically, that it delineates a *category* of people characterised by severe dispositions towards antisocial behaviour (Harris, Rice, and Quinsey 1994; Harris et al. 2007), there are more studies indicating that psychopathy is not a discrete category (Walters et al. 2011). For instance, studies on youth and adults have indicated that psychopathic traits are best construed as dimensional, in the sense that they may be differently pronounced across individuals in the general population, and do not comprise a separate category (Edens et al. 2006, 2011; Murrie et al. 2007). Thus, studies indicate that differences between psychopaths

and non-psychopaths ‘reflect differences in *degree* rather than differences in *kind*’ (Edens, Marcus, and Vaughn 2011, 22).⁴

Other studies indicate that PCL-R’s Factors 1 and 2 are moderately correlated ($r \approx .5$) (Skeem et al. 2011, 101). This gives us a significant probability of finding the co-instantiation of Factor 1 and Factor 2 traits and provides evidence that psychopathy might denote a stable cluster of properties. This is not surprising, since PCL-R was devised to delineate individuals with affective and interpersonal abnormalities *within* the forensic populations that are characterised by antisocial traits as captured by Factor 2 (Hare 2003). However, this also limits its inductive power, because it does not guarantee that these two subsets of psychopathy traits will be reliably co-instantiated in general populations. In fact, researchers indicate that it is likely that affective (e.g. unempathic) and interpersonal traits (e.g. manipulative/conning) can be exhibited without severe antisocial tendencies (e.g. violent and impulsive behaviour; Cooke and Michie 2001).⁵

Nonetheless, it might be argued that psychometric features of PCL-R and its internal reliability capture an SPC kind *within* the population of incarcerated criminals.⁶ We might call this kind prison or criminal psychopaths. However, this suggestion faces the problem of artificially reifying ‘prison psychopaths’ rather than capturing the explanatory potent SPCs that characterise some people (for a discussion, see Jalava, Griffiths, and Maraun 2015, appendix B).⁷ To avoid the illegitimate reification, this proposal should be supplemented with data about how PCL-R correlates with external criteria. Importantly, this includes checking how PCL-R relates to psychometric features of other measures of psychopathy. But it also involves checking whether it predicts something non-trivial and informative about people classified as psychopaths, such as, for instance, their performance on different cognitive, neural and behavioural tasks (Malatesti and McMillan 2014; Brazil 2015). We discuss the latter issue in the next subsections.

Thinking about other measures of psychopathy reminds us that although PCL-R is considered the ‘gold standard’ for measuring psychopathy, it should not be equated with it (Cooke and Michie 2001). In fact, other measures of psychopathy do not provide evidence for the claim that psychopathy is unitary enough to warrant inferences that underpin SPC kinds. For instance, the psychometric studies of PPI indicate that psychopathy is best construed as a ‘constellation of subtraits that are largely or entirely uncorrelated’ (Lilienfeld 2013). Specifically, an important meta-analysis showed that the Fearless/Dominance factor is weakly correlated with other psychopathic traits as measured by PPI (Marcus, Fulton, and Edens 2013). This is important, because FD includes traits ‘such as sensation seeking, narcissism, functional impulsivity, instrumental aggression, and attenuated fear-potentiated startle’ that are ‘theoretically relevant to [different conceptualizations of] psychopathy’ (Lilienfeld 2013, 85). Thus, there is no converging evidence related to different measures of psychopathy for thinking that psychopathy involves a highly correlated cluster of affective, cognitive and behavioural traits.

Given these results, some authors question the supposition that drives psychopathy research, namely, the idea that psychopathy denotes a syndrome or a highly correlated property cluster (Lilienfeld 2013; Jalava, Griffiths, and Maraun 2015, 192–199). On this view, psychopathy should be regarded as a differential set of traits, such as superficial charm, lack of social anxiety, manipulateness and disinhibition, which could be more or less correlated and pronounced across individuals. Since people with some of these traits would be seen as ‘the quintessential social chameleons and social deceivers’, they

would force us ‘to be on the lookout for them’ (Lilienfeld 2013, 86). Some even claim that psychopathy represents a mere instrument of social control of people that we perceive as different from us (e.g. Mullen 2007). On these interpretations, what makes psychopathic traits salient are our characteristic responses, rather than some specific unity underlying these traits.

The lack of high statistical correlation in a measure of psychopathic traits does not necessarily show that psychopathy is not a natural kind. Lack of unity at the level of cognitive, affective and behavioural traits might indicate that an operationalisation of the construct is not valid. For instance, if PPI’s and PCL-R’s items differently correlate with each other and various other measures or experimental tasks, then it is possible that one or the other is not a good measure of psychopathy. Here, the HPC approach to natural kinds could provide a methodological guidance. If psychopathy is a natural kind, we expect to find a set of underlying characteristic causes or mechanisms.⁸ As mentioned above, some authors maintain that psychopathy is underpinned by a specific aetiology. In what follows, we examine this suggestion.

4.2. *The Grounding of Psychopathy*

There are at least two research camps that conceptualise psychopathy as a syndrome underpinned by a common cause. One set of accounts conceptualises psychopathy as underpinned by affective deficits, while the other as involving more general cognitive or information-processing deficits (Brazil and Cima 2016).

According to the affect-based accounts, psychopaths exhibit deficits in the ability to experience and learn from affective stimuli, as exhibited, for instance, on instrumental learning tasks. These tasks involve learning to associate affective valence of stimuli with behavioural responses (Blair, Mitchell, and Blair 2005). Evidence indicates that psychopaths abnormally form associations *via* fear conditioning. In general, studies suggest that psychopaths have problems in recognising fearful and sad stimuli (e.g. related to facial expressions), they show reduced levels of anxiety, startle reflex and other responses to threatening stimuli (Patrick 1994).

These affective deficits prompted David Lykken (1995) to propose the Low Fear model of psychopathy. According to this model, psychopathy is marked by a general deficit in responding to fearful stimuli. Later developments of this model include James Blair and colleague’s postulation of an impaired Violence Inhibition Mechanism (VIM; Blair, Mitchell, and Blair 2005). These deficits in responding to affective stimuli could explain why psychopaths engage in immoral and antisocial behaviour. For instance, deficits in recognising sad and fearful cues might not activate VIM that in normal people inhibits violent and aggressive behaviour. Also, deficits in recognising fearful stimuli might explain why psychopaths do not learn from punishment and persevere in harmful behaviour despite its negative consequences.

However, fear-based models of psychopathy have been criticised as oversimplifying affective deficits correlated with psychopathy (Brazil and Cima 2016, 212). Studies indicate that fear is a multifaceted construct and that psychopaths might not be subjectively insensitive to fearful stimuli that lead to punishment; instead, they might simply fail to detect such information (for a review, see Hoppenbrouwers, Bulten, and Brazil 2016). Thus, Blair (2005) has proposed a more general affect-based account of psychopathy, the so-called

Integrated Emotion Systems (IES) model. According to IES, ‘psychopathic behaviour is driven by impairments in *representing* affective information, thus leading to disturbances in specific associative learning processes’ (Brazil and Cima 2016, 215, emphasis added).

Some neurocognitive data support this view. It has been shown across studies that the PCL-R scores correlate with abnormal functioning of brain regions that process affective stimuli and underpin reinforcement learning. These include the amygdala, ventromedial (VMPFC) and orbitofrontal (OFC) prefrontal cortex, anterior cingulate cortex and larger paralimbic area of the brain (Kiehl 2006; Blair 2008).

The information-processing accounts are based on the discovery that psychopaths’ behavioural and affective deficits are context-dependent, namely their manifestation depends on the focusing of psychopaths’ attention. According to the Response Modulation Hypothesis and its variants advanced by Joseph Newman and colleagues (e.g. Koenigs and Newman 2013), functional deficits in psychopathy stem from a failure to reallocate attention from a salient cue that is in their focus, to a secondary cue, that is outside of their focus, but might be relevant for successful accomplishment of a task. In addition, Newman and colleagues have shown that deficits in reinforcement learning are exhibited among psychopaths even when they are required to respond to neutral (non-emotional) stimuli, indicating that the deficits are not restricted to affective stimuli. However, when their attention is properly directed, abnormalities correlated with fear-potentiated startle reflex, passive avoidance learning, emotion recognition, and the amygdala activation tend to disappear (e.g. Newman and Kosson 1986; Koenigs and Newman 2013; Larson et al. 2013).

These two research paradigms are standardly presented as offering alternative causes of the psychopathy syndrome (Hamilton, Racer, and Newman 2015). We do not adjudicate between the two accounts. Both accounts are developed on an impressive amount of research. However, we will indicate why the currently available evidential basis for either of the accounts does not provide sufficient evidence for considering psychopathy as a natural kind.

Most evidence for these accounts is based on the results from tasks that tap different aspects of executive functions (EF; Koenigs and Newman 2013). EF refer to capacities such as planning, attention, memory, response control and so on that underlie decision-making. However, different studies gave inconsistent results regarding psychopaths’ performance on these tasks. A plausible explanation of the inconsistent results is the fact that psychopathy is a constellation of loosely correlated traits, and thus not a unified syndrome (Lilienfeld 2013).

The affect-based accounts mostly rely on evidence from psychopaths’ performance on the so-called hot executive function tasks that tap brain structures that process motivationally salient stimuli (Maes and Brazil 2013). A paradigmatic example is the Iowa gambling task, devised by Bechara and colleagues to test the Somatic Marker Theory (Bechara et al. 1994; Damasio 1994). The idea is that people with normally functioning somatic markers will spontaneously acquire affectively negative representations when making disadvantageous decisions and learn to associate positively valenced representations with the advantageous decisions. Some studies indicated that psychopaths might have deficits in affectively marking the relevant information (for a review, see Blair, Mitchell, and Blair 2005). However, other studies have failed to replicate these results on the Iowa gambling task (Schmitt, Brinkley, and Newman 1999; Blair and Cipolotti 2000; Lösel and Schmucker

2004). Similar inconsistency or mixed results have been found in other studies that tap hot EF *via* various classical or operant conditioning methods and those that used imaging technology (e.g. Baskin-Sommers, Curtin, and Newman 2011, 2013; Larson et al. 2013).

Some of these incongruities can be explained by the attention-based accounts, such as the response modulation hypothesis and its variants (Koenigs and Newman 2013). These accounts predict situation-specific deficits in processing affective stimuli. Affective deficits are expected when affective stimuli are not in psychopaths' primary focus of attention. However, the evidence for the attention-based accounts does not support the view that psychopathy is a natural kind. Specifically, some studies have failed to confirm the predictions of attention-based accounts related to psychopaths' cognitive functioning (Zeier et al. 2012). Moreover, what might account for this limited predictability is the fact that attention moderates affective deficits only in *primary* psychopaths (Zeier, Maxwell, and Newman 2009; see also Schultz et al. 2016). Primary psychopaths are those who score high on PCL-R but show low anxiety traits. Alternatively, secondary psychopaths show high anxiety traits. Thus, many proponents of the attention-based accounts accept the view that psychopathy is not a unitary construct (Zeier, Maxwell, and Newman 2009, 2012; Schultz et al. 2016).

Furthermore, a more general explanation for the incongruity in the data is that decision-making deficits differentially correlate with *specific* psychopathy traits. This would be expected if 'psychopathy' denotes a loosely correlated set of features. This contention seems to be confirmed across studies. For instance, Dean and colleagues (2013) found that in a general population, risky decision-making on the Iowa gambling task is significantly correlated with impulsive and volatile personal style, while it is not correlated with the callous and unemotional personality traits. This indicates that psychopathy is not a monolithic entity underpinned by a clearly discernible set of dysfunctional mechanisms.

Baskin-Sommers and colleagues (2015) provide more general evidence for the latter hypothesis. On the supposition that psychopathy is not 'a broad clinical syndrome', they investigated how psychopathy facets relate to executive function as measured by a battery of EF tasks. The results show that the PCL-R Factor 1 is not significantly correlated with EF, while Factor 2 is negatively correlated with EF performance. Specifically, the anti-social facet of Factor 2 seems to be negatively correlated with EF performance. This indicates that Factor 1 traits might have a different aetiology from Factor 2 traits (Baskin-Sommers et al. 2015, 337). If our construct of psychopathy denotes a natural kind, these results would be harder to explain. However, they are easily explained if psychopathy is a dimensional constellation of traits with different causal histories that can be more or less pronounced across individuals.

4.3. Neurobiological Markers of Psychopathy

It could be argued that the diagnostic biomarkers of psychopathy indicate the existence of a unitary construct. Biomarkers include all objectively measurable factors, such as physiological reactions, gene sequences, specific brain activation patterns, and so on, that are used for detecting, diagnosing and predicting biologically normal and abnormal conditions. Specifically, the EEG, PET scan, and fMRI studies found that psychopathy correlates with abnormalities in the activity of brain areas, such as the ventromedial and

orbitofrontal prefrontal cortex, parts of the anterior cortex, insula, amygdala and the functional connections between them (e.g. Glenn and Raine 2014).

These findings prompted some researchers to speculate that there is ‘a diagnostic biomarker’ for psychopathy (Gregory et al. 2015, 154). However, biomarkers do not have a high predictive value on their own; they need to be coupled with genetic and developmental data to provide explanatory and predictive amendments (Brazil 2015, 116). This complicates the classification of psychopathy and arguably indicates a need to refine the category.

For instance, there is evidence that the core psychopathic traits pertaining to affective and interpersonal deficits are moderately to highly heritable (Viding et al. 2008; Glenn and Raine 2014, 23). However, recent evidence indicates that the heritability of affective and interpersonal traits of psychopathy explains specific brain patterns related to antisocial and criminal behaviour via the mediation of specific environmental circumstances. Kolla and colleagues (2013, 2014) discovered a significant interaction between antisocial aspects of psychopathy and childhood physical abuse (CPA). Furthermore, Dargis and colleagues (2017) indicate that the correlation between psychopathy and deficits in reinforcement learning is moderated by CPA. These studies show that familiar correlates of psychopathy, such as brain abnormalities, functional impairments and antisocial behaviour, are exacerbated by the environmental conditions. In that regard, the biomarkers of psychopathy can only play explanatory and predictive roles on the background of this interaction between the heritable psychopathic traits and adverse childhood conditions.

This point can be nicely illustrated with the case of neuroscientist James Fallon. Fallon discovered that PET scan images of his brain show surprisingly similar activation patterns with the brains of psychopathic criminals. This included a diminished activity in the circuitry between orbital and ventromedial prefrontal cortex, cingulate cortex and the amygdala (Fallon 2013, ch. 3). Fallon and his family were not entirely surprised by the results. From childhood, Fallon has shown callous, unempathic, manipulative traits and proneness to risky behaviour. Nonetheless, he has not exhibited antisocial traits. For example, on PCL-R, he scores high on Factor 1, but low on Factor 2 traits and on PPI, he scores high on the FD traits. Quite the opposite from a typical psychopath, Fallon is a family man and a successful and respected neuroscientist. Thus, if sole reliance on brain patterns provided diagnostic biomarkers of psychopathy, then we would group together a disparagingly different types of people. This category would have little explanatory and predictive power, which would make psychopathy a poor candidate for a natural kind on any of the proposed accounts.

It could be argued that Boyd’s HPC can accommodate these considerations. As mentioned in the introduction, HPC can countenance kinds that are underpinned by external or extrinsic mechanisms including environmental factors. Thus, the claim could be that psychopaths comprise a group of people who have characteristic biomarkers that are underpinned by gene–environment interactions.⁹

However, this proposal will not apply to the current conceptualisation of psychopathy. The reasons for this are related to the already noted heterogeneity and lack of consistency between different measures of psychopathy and how they correlate with external criteria, such as performance on different neuropsychological tasks and measures of biocognitive functions. To illustrate, let us consider again studies that utilise the distinction between primary and secondary psychopathy. The distinction

itself (low/high anxiety) indicates a difference in biomarkers underpinning psychopathic personality. Moreover, studies that utilise this distinction found differential neurocognitive signatures between high- and low-anxious psychopaths (e.g. Zeier, Maxwell, and Newman 2009; Koenigs, Kruepke, and Newman 2010; Schultz et al. 2016). To take one important example, in a recent study, Schultz and colleagues (2016) indicate that during classical fear conditioning, primary psychopaths show normal activation in the dorsal cingulate cortex and reduced activation in the ventral cingulate cortex, while secondary psychopaths showed an inverse pattern of activation in those brain areas. This indicates that the mechanisms underpinning fear conditioning, which according to some accounts present a hallmark of psychopathy (Blair, Mitchell, and Blair 2005), might be intact in primary psychopaths, but impaired in secondary psychopaths. In effect, the differences in the brain activation and performance of primary and secondary psychopaths on various cognitive and conditioning tasks ground different predictions and explanations of their cognitive, affective and behavioural capacities. Moreover, they indicate that strategies for controlling such behaviour and designing therapies for remedying these abnormalities should be different, depending on the underpinning impairments or abnormalities (Brazil et al. 2016). Thus, these results provide evidence that psychopathy as measured by PCL-R and other tools is not best construed as a natural kind on the HPC family of accounts.

5. What to Do with Psychopathy? Some Methodological Remarks

Our discussion supports the so-called *clutter hypothesis* about the construct of psychopathy. According to Matteo Mameli, a construct is ‘cluttered’ when it ‘conflates different properties, properties that, according to our current best theories, need to be kept distinct’ (Mameli 2008, 720). To use a well-known example, once it was thought that jade was a natural kind. Now we know that ‘jade’ refers to two microstructurally different kinds of things (jadeite and nephrite) whose different properties warrant different inferences about their instances. Mameli introduces the clutter hypothesis in relation to the concept of innateness which seems to conflate properties such as being a genetically coded Darwinian adaptation, having a flat norm of reaction, being universal within a species, and so on. A general problem with such heterogeneous constructs is that they may ground incompatible inferences and classifications (Mameli 2008, 731–732).

As the reviewed research indicates, psychopathy has different aetiologies and biocognitive correlates and it is even differently conceptualised and measured across different research groups (Brazil et al. 2016). When a research domain contains a ‘cluttered’ construct, it would benefit from introducing some changes related to that construct (Mameli 2008). Some authors agree that this point applies to the psychopathy research (Brazil et al. 2016). In general, however, it is not immediately clear what needs to be changed.

In what follows, we consider three possible responses to the general results of our discussion. First, we might revise the category of psychopathy. Second, and perhaps following from the first response, the current category ought to be eliminated due to aforementioned difficulties associated with it. Third, it could be argued that although psychopathy is not a kind on cluster approaches, there are more suitable accounts of kinds in the biomedical sciences that should replace the cluster approaches and that can countenance psychopathy as a kind. We start with the third response.

5.1. *Revising the Account of Kinds*

Recently, some authors have analogously to our discussion argued that autism is not an HPC kind (for a review, see Weiskopf 2017). In response to these considerations, Daniel Weiskopf (2017) argues that autism should not be judged by the HPC or similar criteria. According to him, autism is a *heterogeneous* kind best captured by a network model of kinds. In a networked category, one starts with a set of idealised exemplars grounded on individual cases that form a clinical profile. The exemplars form a network by sharing at least one theoretically significant property with exemplars from other sets.¹⁰ The significant property might refer to biomarkers, behavioural dispositions, functional and cognitive profiles, and so on. Similarly, it could be argued that we should not apply the natural kind framework to psychopathy, rather we should conceptualise the category in some other way.

Without entering into debate on the specific case of autism classification, we take it that Weiskopf's approach, if it pertains to displacing natural kinds altogether from the biomedical sciences, is not a promising strategy in general, and more specifically in the case of psychopathy. We provide three interrelated considerations why abandoning the standard natural kind framework is not a promising strategy for handling psychopathy.

First, the function of kinds in the biomedical sciences is to provide us with explanatory and predictive information that grounds control and effective treatments. Cluster approaches pertain to accommodate this function. Alternative accounts either capture these explanatory and predictive relations or they do not. If not, they lack something important as accounts of categories in the biomedical sciences; namely, they do not capture relations that ground knowledge necessary for effective control and treatment. If they capture these relations, then they will not advance the current debate because psychopathy will likely not be a kind for the already presented reasons.

Second, as mentioned above, important goals in the biomedical sciences involve creating categories that facilitate successful diagnosis and prediction of pathological processes and designing therapies for curing them. However, the scientific research on psychopathy and ASPD more generally has not been successful in designing effective therapies (Brazil et al. 2016). Given this stage of the current research, it is unlikely that abandoning the natural kind framework and revising the account of kinds in order to countenance psychopathy as it is currently conceptualised will improve the prospects of designing better therapies. This leads us to the third point.

Philosophical accounts of natural kinds should not just accommodate and describe currently used scientific categorisations; they should also provide a normative framework for judging what counts as a natural kind and when a category should be revised or eliminated (Kendler, Zachar, and Craver 2011). Otherwise, we run the risk of legitimising current classifications, even when they hinder scientific progress. Property cluster approaches provide a framework for avoiding such illegitimate conservatism. These three considerations indicate that the psychopathy research might be improved by revising the category of psychopathy, rather than abandoning the natural kind framework.

5.2. *Revising the Category of Psychopathy*

In the literature, there are some indications how the constructs of psychopathy and ASPD could be revised into finer grained types. As already discussed, some authors propose to

distinguish between primary and secondary psychopathy. There are different but inter-related dimensions along which authors propose to introduce this distinction (Yildirim and Derksen 2015). As mentioned, often the distinction is made in terms of levels of anxiety. Different levels of anxiety might reflect differences in aetiology (Schultz et al. 2016). For instance, Benjamin Karpman (1941) speculated that primary psychopaths have an *inherited* affective deficit while secondary psychopaths have an *acquired* affective disturbance (see also Skeem et al. 2003). There is even some evolutionary evidence supporting this distinction. Affective and interpersonal (Factor 1) traits seem to be positively correlated with reproductive success among psychopathic offenders, indicating that these traits might be heritable because they play a protective role for people growing up in adverse environments (Međedović et al. 2017). Nonetheless, research shows that the distinction between primary and secondary psychopaths can be made along different neuropsychological and behavioural dimensions and that further subtypes can be distinguished within these posited categories (for a review, see Yildirim and Derksen 2015). Thus, the usefulness of this distinction and its validity should be further evaluated by future research.

Other authors argue that for practical and theoretical reasons, we should distinguish between successful and unsuccessful psychopaths (e.g. Sifferd and Hirstein 2013). Researchers are still not clear on how exactly to conceptualise the distinction (for a review, see Smith, Watts, and Lilienfeld 2014). However, there is a broad understanding that successful psychopaths would be intelligent individuals displaying abnormal affective and interpersonal traits (Factor 1), but with no or little history of violent behaviour and incarceration. The unsuccessful psychopaths would be those who also display the anti-social and impulsive traits (Factor 2). However, the construct of successful psychopathy and its measurement are still not validated, and many claims about the existence of successful psychopaths who have superior cognitive skills seem to rest on dubious studies (Maes and Brazil 2013; Jurjako and Malatesti 2016).

However, if 'psychopathy' designates a loose set of traits that do not form a reliable and stable cluster, as seems to be suggested by Lilienfeld (2013), then the prospects of discovering subtypes that would be analogous to the case of jade are not promising. In that case, psychopathy research could benefit from a more radical revision. Given that there is no consensus on what are the core psychopathic traits and how to measure them and that we have no effective therapies for the most severe forms of ASPDs, Inti Brazil and colleagues (2016) suggest to reconceptualise all antisocial personality types along the lines of the Research Domain Criteria (RDoC) project. The National Institute of Health proposed RDoC as a way of reconceptualising the classification of mental disorders (Insel et al. 2010). RDoC conceptualises mental disorders as dysfunctions in the brain circuits and relies on genetic and other neuroscientific data to provide biocognitive markers for diagnosing and classifying disorders (Insel et al. 2010; see also Murphy 2017).

This way of classifying might be fruitfully applied to psychopathic and antisocial behaviour. The reviewed studies indicate that different factors cause or correlate with antisocial behaviour, including psychopathy. The neurocognitive biomarkers, including the heritability of some psychopathic traits, might delineate novel specific groupings within the broader category of ASPD. When these biomarkers are coupled with information about the early developmental environment and other relevant factors, they may provide reliable information about different subtypes of people exhibiting severe forms of antisocial behaviour and the causes or mechanisms underpinning that behaviour. The suggestion of Brazil

et al. (2016) offers an alternative way of categorising that would not depend on different and historically contingent folk-psychologically defined measures of psychopathy. Furthermore, this information about developmental and biological markers of psychopathy and ASPD might be used for developing more focused and effective treatments for people with extreme forms of ASPD (Brazil et al. 2016). This bottom-up reconceptualisation could be an alternative way of unravelling the natural kind that our construct of psychopathy might be gesturing at.

Reorientation to bottom-up research, however, might suggest the elimination of the construct of psychopathy. This brings us to the first proposed response—to eliminate the category. Although we leave open that in future the construct of psychopathy will become theoretically and/or practically obsolete, we maintain that this is still not the case. In the next section, we indicate how we think the role of ‘psychopathy’ should be construed in the current research.

6. Psychopathy as a Pragmatic Kind

Although the clutter hypothesis seems to apply to the construct of psychopathy, it does not follow that the category is entirely practically or theoretically useless (but see Blackburn 1988). For instance, Mameli thinks that the clutter hypothesis is true of Newton’s concept of mass, since it conflates rest mass and relativistic mass (Mameli 2008, 730). However, since they simplify calculations, Newton’s concept of mass and equations that define it are still useful for some purposes, such as building bridges. Albeit relevantly different from the concept of mass, we maintain that psychopathy as currently used is useful for some research purposes and thus, should not be eliminated.

We maintain that a category is minimally useful, and thus a pragmatic kind, when it focuses research by facilitating communication and prediction in some domain.¹¹ Psychopathy satisfies these conditions in some research domains. For instance, psychopathy, at least as characterised by PCL-R, reliably predicts recidivism and violent behaviour (Hare 2003).¹² Thus, it plays an important practical role in legal and clinical contexts where it is often important to predict dangerousness of a person (Malatesti and McMillan 2014; Brzović et al. 2016). Furthermore, in forensic research, it informatively carves up a subgroup of people with ASPDs. For instance, studies indicate that people with ASPD comprise 50% of prison populations across countries (Fazel and Danesh 2002), while only 20% of those populations is considered psychopathic (Hare 2003). Thus, psychopathy narrows down the scientific focus to groups of people within the broader category of ASPD. Also, psychopathy has an important communicative role in forensic and clinical research. Many of the most important discoveries regarding psychopathy were found after the standardisation of assessment procedures such as PCL-R. There is an impressive body of information stemming from the neuropsychological research on psychopathy which might be lost if we discard ‘psychopathy’. We think that these considerations qualify psychopathy as an important pragmatic kind.

The concept of a pragmatic kind, as we construe it here, is useful to accommodate situations where a category cannot be considered a natural kind, but it serves useful investigative purposes at least until a better alternative becomes available. Most scientific categories are introduced as pragmatic kinds in this sense. Take the concept of a gene, for example. It was introduced as a vague notion of a unit of inheritance. Nonetheless, it serves a unifying

role in scientific research that enabled discoveries of mechanisms underpinning genetic material and its causal roles (Rheinberger 1997). Thus, while theoretically 'gene' refers to a wide and heterogeneous class, for practical purposes, it is still useful.¹³

This approach to psychopathy seems to be vindicated by the Triarchic model of psychopathy. Recently, Patrick, Fowles, and Krueger (2009, 913) have proposed the Triarchic model as 'a framework for coordinating research on neurobiological and developmental processes contributing to varying manifestations of psychopathy. This model is based on a review of different historical and contemporary conceptualisations of psychopathy. It integrates competing and inconsistent conceptualisations and empirical results by conceptualising psychopathy as comprised of three broad features that have different aetiologies and can be more or less displayed across individuals. These include Disinhibition (e.g. impulsiveness, control problems), Boldness (e.g. low reactivity to stress, high self-assurance) and Meanness (e.g. exploitative and cruel behaviour; Patrick, Fowles, and Krueger 2009, 925–926).

We maintain that the reviewed considerations qualify psychopathy as an important case of a scientific category in the making, where a category, regardless of its deficiencies, serves to guide further research. Thus, we think that according to the available research, psychopathy should be construed as a pragmatic kind that underpins forensic and clinical research on extreme forms of antisocial behaviour, at least until a better refined construct or some other theoretically and practically more useful concept replaces it. Indeed, we expect the category to be revised and refined in future research for at least one important reason. As mentioned in section 5, currently, there is no effective (cognitive, behavioural or pharmacological) therapy for reducing the maladaptive features of psychopathy (Brazil et al. 2016). This is likely due to the heterogeneity of psychopathy which underlines our argument that psychopathy is not a natural kind. We expect that a likely remedy for this important problem will include revisions along the lines we discussed in the previous section.

7. Conclusion

We examined whether psychopathy can be considered a natural kind on the cluster approaches. We argued that currently there is insufficient evidence to conclude that psychopathy comprises a stable cluster of properties and, furthermore, that studies suggest that psychopathy is not underpinned by a unified set of underlying mechanisms. Subsequently, we considered implications of such results for the category of psychopathy. Three options were examined: (1) revision of the category; (2) elimination of the category and (3) abandoning the cluster approach to natural kinds. Against (3), we argued that the cluster approach to psychopathy categorisation should not be abandoned. In our view, the scientific, forensic and clinical practice would benefit from the revision of the psychopathy construct. However, we maintain that this does not imply that the construct should be eliminated because it still plays useful roles in framing research. To distinguish its current status from categories that designate natural kinds, we propose to construe 'psychopathy' as a pragmatic kind.

Notes

1. In Table 1, item numbers denote their original sequence in the PCL-R manual.

2. Since the number of psychopaths in the European prisons is smaller, the cut-off line of 25 provides the equivalent psychometric features as the score 30 in North America (Cooke and Michie 1999; but see Hare 2003, 30).
3. However, there are versions of PCL devised for younger populations (PCL-Y) and for research in non-institutional settings (PCL-screening version) (Hare 2003).
4. This is not surprising since studies indicate that most disorders exhibit a dimensional and thus non-taxonic structure (Haslam, Holland, and Kuppens 2012).
5. A nice example of this dissociation between Factor 1 and Factor 2 scores is provided by the case of Dr. Fallon. See section 4.3.
6. Thanks to an anonymous reviewer for pressing us to explicitly address this issue.
7. Interestingly, given the problem of reification, some researchers informally call psychopaths identified by PCL-R as Bob Hare's psychopaths.
8. This characterisation of HPC might imply that all members of the kind psychopathy need to share common causes responsible for their shared properties. An anonymous reviewer rightly points out that this is not necessarily Boyd's view, although this seems to be a common interpretation of his account (Slater 2015). Arguably, Boyd's view is compatible with Slater's stable property cluster account. In fact, Slater (2015) accepts the possibility that his view represents only a minor revision to Boyd's account. However, in the present context, the correct interpretation of Boyd's view is non-essential. We only emphasise the fact that there are important differences between these two views (or interpretations of Boyd's account) that have practical consequences for identifying psychiatric and forensic categories such as psychopathy.
9. Thanks to an anonymous reviewer for raising this issue.
10. This view of kinds should not be confused with a more familiar network theory of mental disorder (Borsboom 2017). This theory requires stable clustering of causally self-sustaining symptoms to determine a disorder. In this respect, it is compatible with clustering approaches to kinds.
11. The term 'pragmatic kinds' originates in Philip Kitcher's (2007; see also Zachar 2002) approach to natural kinds in general. He takes kinds to be those categories that best serve the goals our community would endorse in an informed and educated democratic decision process. We do not endorse such an approach to all scientific categories. However, we hold that his strategy of weighing practical and epistemic considerations is suitable for the scientific contexts where we still do not have well-established categories, but only certain open-ended attempts, or categories in the making. In the main text, we explain how these considerations apply to the current stage of psychopathy research.
12. However, the predictive power of PCL-R is mostly due to antisocial traits which psychopathy shares with more general antisocial personality disorder (Wallinius et al. 2012).
13. Paul Griffiths (2004), following Ingo Brigandt (2003), uses the notion of investigative kinds to capture similar ideas about some scientific categories (see also Rheinberger 1997). Brigandt (2003) construes investigative kinds as scientific concepts in the ongoing research projects whose intensions and extensions are altered through research to achieve greater explanatory and predictive power. We do not adopt the term 'investigative kind' because Brigandt (2003, 1309–1310) seems to construe it as a category that loses its purpose once discovered that its instances do not share an underlying mechanism or a structural property. Thus, 'investigative kinds' seem to be a terminological variant of natural or cluster kinds. Instead, we use the concept of pragmatic kinds to delineate exactly those stages of scientific research where the categories are not yet fully established or determinate but are useful in the ongoing research.

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